DECLARATION OF PAUL BASS, PH.D.

I, PAUL BASS, PH.D., have been retained as an expert on behalf of Corepharma, LLC ("Corepharma") to assess the pharmacokinetic results of certain clinical studies conducted in connection with the muscle relaxant metaxalone, which is marketed under the trade name Skelaxin®. I have also been retained to assess the safety and efficacy of such results and the impact of excluding such results from the labeling for generic metaxalone products.

I. STATEMENT OF QUALIFICATIONS

1. I was awarded the degree of Bachelor of Science in Pharmacy in 1953 and the degree of Master of Arts in Pharmacology in 1955, both from the University of British Columbia. I was awarded the degree of Doctor of Philosophy in Pharmacology from McGill University in 1957.

2. Following receipt of my doctoral degree, I spent one year as a postdoctoral student in the Departments of Physiology and Biochemistry at McGill University, and then spent 18 months as a fellow in the gastrointestinal section of the Department of Physiology at the Mayo Foundation.

3. My postdoctoral training was sponsored by Parke, Davis and Company, and following its completion, I assumed the position of Research Pharmacologist at Parke, Davis. I was subsequently promoted to Senior Research Pharmacologist in 1967 and then Associate Laboratory Director in 1968.

4. Concomitant with my employment at Parke, Davis, I was a Lecturer in the Department of Pharmacology at the University of Michigan from 1966-1970.

5. I have been on the faculty of the University of Wisconsin – Madison since 1970, with a dual appointment as a Professor in the School of Pharmacy, and a Professor of Pharmacology in the School of Medicine. From 1980-1984, I was also a Professor of

Pharmacology in the School of Veterinary Medicine. I am currently an Emeritus Professor of the University of Wisconsin Schools of Pharmacy and Medicine.

6. My research interests include the effect of drugs on the gastrointestinal tract, including the absorption of drugs and the control of stomach emptying. In addition, I am interested in clinical trials of drugs affecting both the gastrointestinal and pulmonary systems.

7. I am a member of several professional societies and organizations, including the American Society for Pharmacology and Experimental Therapeutics, and the American Gastroenterological Association.

8. I have been a member of the Editorial Board of the *American Journal of Physiology;* the *Journal of Pharmacology and Experimental Therapeutics;* the *Journal of Pharmacological Methods;* and *Neurogastroenterology and Motility.* I currently review publications for several journals, including the *Journal of Medicinal Chemistry* and *Gastroenterology.*

9. I have published over 120 research publications, 27 chapters and reviews, and 87 abstracts in the area of the gastrointestinal tract. I have given more than 35 invited presentations, and have received over 30 grants and awards for conducting gastrointestinal pharmacological research, including numerous grant awards from the National Institute of Health.

10. A copy of my Curriculum Vitae is attached to this Declaration as Appendix A.

II. OVERVIEW OF OPINIONS

11. I understand from the March 1, 2004 letter from the Food and Drug Administration (FDA) Office of Generic Drugs regarding "ANDA for Metaxalone Tablets" that the FDA will approve labeling for generic metaxalone that "carves out" the fed-state bioavailability data currently included in the labeling for Skelaxin®. I have also read the Citizen Petition submitted to the FDA by King Pharmaceuticals, Inc. ("King"), and the exhibits thereto.

King's Citizen Petition objects to the omission of this information from generic metaxalone labeling, and further proposes that the FDA include certain additional pharmacokinetic data regarding the effects of age and gender on the bioavailability of metaxalone, as well as an instruction to administer metaxalone with food to minimize the age effect that King reports.

12. I have been asked to opine regarding whether the food-effect information to be carved out of the labeling for generic metaxalone has any clinical relevance with respect to the safe or effective use of metaxalone, and whether the omission of that information from generic metaxalone labeling would render the drug less safe or effective. I have also been asked to opine regarding whether the age-effect or gender-effect information cited in King's Citizen Petition, as well as the food instruction that King proposes, have any clinical relevance with respect to the safe or effective use of metaxalone, and whether the omission of this information from generic metaxalone labeling would render it less safe or effective.

13. In my opinion, the food-effect information to be carved out of generic metaxalone labeling is not clinically relevant to its safety or efficacy, and the omission of that information would not render generic metaxalone less safe or effective.

14. As explained more fully below, because the mechanism and exact location of action of metaxalone are unknown, and due to the lack of information correlating safety or efficacy of the drug with plasma concentration levels, in my opinion, the information relating to any differences between fed and fasted blood levels of metaxalone included in the current labeling for Skelaxin is not clinically relevant.

15. Furthermore, in my opinion, when metaxalone is administered as recommended (800 mg t.i.d. or q.i.d.) in conjunction with ordinary food consumption (as opposed to a high-caloric, high-fat breakfast), the difference between fed and fasted steady-state plasma concentration levels would be of an insignificant magnitude or non-existent.

16. In addition, in my opinion, there is no link between the pharmacokinetic information to be carved out of generic metaxalone labeling and any increase in side-effects or other adverse reactions related to the drug. Among other things, I note that daily does of 2,400 to 3,200 mg have been administered safely and effectively for over forty years both with and without food, this recommended dosing schedule has never required adjustment in either case, doses as high as 9,600 mg have been shown safe and effective, and the drug was used safely and effectively for forty years without regard to or notice of any food effect. In view of these facts, there is no suggestion that the increases in bioavailability cited by King would have any bearing on the safety or efficacy of the metaxalone.

17. It is also my opinion based on metaxalone's long and safe history of use as an adjunct with other therapies that any additional drug-drug interactions relating to metaxalone other than those discussed in its current labeling are unlikely. In addition, even if any additional drug-drug interactions should occur, the food-effect information cited by King would not assist in their determination.

18. For these reasons, as more fully explained below, it is my opinion that the fed-state bioavailability data that the FDA will allow to be carved out of the labeling for generic metaxalone has no clinical relevance to the safe and effective use of metaxalone, and that the omission of this information from generic metaxalone labeling would not render the drug less safe or effective.

19. For similar reasons, it is also my opinion that the age-effect and gender-effect information data cited by King is also clinically irrelevant, that the omission of this information also would not render generic metaxalone less safe or effective, and that this information does not warrant the food instruction that King proposes.

III. EXPERT OPINIONS

A. Physiology of the Human Gastrointestinal Tract

20. The stomach is the first enlargement of the human gastrointestinal tract to receive various ingested nutrient and non-nutrient material, such as tablets. The emptying of the gastric contents of the stomach is primarily controlled by the duodenum and determined by the chemical composition as well as the physical form of the material in the stomach.

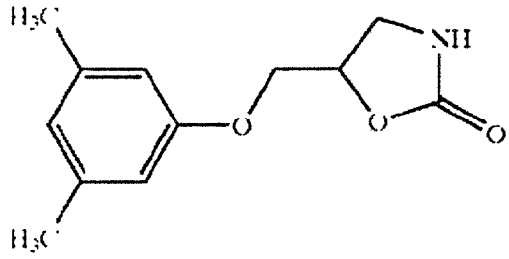
21. For example, water has a gastric emptying half-life of approximately 10 minutes. On the other hand, it is well known that ingestion of a high-fat food will delay gastric emptying by causing contractions of the proximal portion of the small intestine (the duodenum). This delay in gastric emptying allows for powerful contractions in the antrum portion of the stomach to push solids back into the gastric body portion of the stomach. The propulsion (towards the duodenum) and retropulsion (towards the gastric body) by the antrum of the gastric contents increase the residence time of the gastric contents and are responsible for the particle size reduction of solids in preparation for gastric emptying.

22. It has long been widely known that the consumption of a high-fat food with a tablet dosage form containing a hydrophobic and/or lipophilic active pharmaceutical ingredient, such as metaxalone, will typically increase the dissolution of the tablet. Among other reasons, this is due to an increase in the residence time of the tablet in the stomach coupled with propulsion and retropulsion of the gastric contents in the high-fat environment. In addition, after sufficient agitation, the gastric contents are then emptied into the duodenum. Lipophilic compounds are further solubilized in the duodenum by the increased secretion of bile salts induced by consumption of a high-fat meal. Bile salt secretion assists in the formation of micelles which enhance absorption from the small intestine of the lipophilic active ingredients.

23. It has also long been well known that the fasted-state motility of the human digestive system goes through a three-phase propagative pattern known as the migrating motor complex (MMC) every 90 minutes. In Phase I of the MMC, which lasts approximately 60 minutes, very little motor activity occurs. Phases II and III, which each last approximately 20 minutes and 10 minutes respectively, are marked by nearly continuous motor activity capable of sweeping a solid dosage form along with accumulated mucus and secretions into the duodenum. Phase III is also marked by bile secretion into the duodenum. Thus, in addition to bile secretion being induced by the consumption of a high-fat meal, bile is also secreted approximately every 90 minutes (each MMC cycle) into the duodenum in the fasted state. These phenomena are more fully discussed in the article entitled "Gastric Emptying: Differences Among Liquid, Fiber, Polymer and Solid Dosage Forms of Medications." Ex. 24.[1]

B. Absorption of Metaxalone

24. Metaxalone has the following chemical structure:



25. The molecular structure reveals that metaxalone does not have any hydrophilic functional groups, such as hydroxyl or carboxylic acid, indicating that it will have very low water solubility. This lack of water solubility is also confirmed by literature stating that

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[1] All citations are styled as "Ex. __" and refer to the Exhibits submitted with Corepharma's letter of April 30, 2004 regarding its Comments on the Citizen Petition of King Pharmaceuticals, Inc. ("King").

metaxalone is practically insoluble in water. *See, e.g.*, Ex. 3 at 518; Carroll, "The pharmacology of a new oxazolidinone with anticonvulsant, analgetic and muscle relaxant properties," *Arch. Int. Pharmacodyn.*, Vol. 80, No. 3-4:280-98 at 280 (1962) (Ex. 13).

26. The chemical structure of metaxalone also shows the presence of a benzene ring, the heterocyclic moiety, and an ether linkage, all of which are well known indicators of lipid solubility. The lipid solubility of metaxalone is further confirmed by literature stating that metaxalone is freely soluble in chloroform, *see,* Osol, *Remington's Pharmaceutical Sciences* at 867 (16th ed. 1980) (Ex. 15), and can be crystallized from ethyl acetate. *See* Monograph No. 5838 of the Merck Index at 933 (11th ed., 1989) (Ex. 16).

27. As noted above, it is well-known that co-administration of hydrophobic and lipophilic drugs with a high-fat meal will likely increase the bioavailability of the drug. Among other things, the consumption of a high-fat meal increases the residence time of the meal and tablet in the stomach, stimulates propulsive and retropulsive motions of the gastric contents and leads to the secretion of bile salts that aid in micelle formation and increase drug absorption. Additionally, fasted-state bioavailability can also be increased if the administration of metaxalone corresponds with the fasted-state secretion of bile into the duodenum every 90 minutes associated with Phase III of the MMC. *See*, Hamaguchi, "Effect of a high-fat meal on the bioavailability of phenytoin in a commercial powder with a large particle size," *Int'l. J. Clin. Pharmacol., Ther., and Tox.*, Vol. 31, No. 7:326-33 (1993).

28. This resulting increase of absorption in fasted-state administration of metaxalone is reflected in data that appears in United States Patent Nos. 6,407,128 (the '128 patent) and 6,683,102 (the '102 patent). Exs. 17 & 20. In Table I of both patents, the ratio of plasma concentration levels in the fed and fasted conditions (Ratio A/B) is less than 1.00 in 10 out of 44 subjects. It is my opinion that the explanation for these 10 subjects exhibiting an increase in

plasma concentration levels in the fasted state versus the fed state is due to the fasted administration of metaxalone at a time corresponding to the secretion of bile into the duodenum associated with Phase III of the MMC. This data thus reflects that plasma concentration levels of metaxalone in the fasted state can exceed the levels in the fed state when the fasted-state administration corresponds with the secretion of bile into the duodenum associated with Phase III of MMC.

C. The Food Effect Cited by King is Clinically Irrelevant to the Safety and/or Efficacy of Metaxalone.

29. In my opinion, there is no established relationship between the safety or efficacy of metaxalone and the plasma concentration levels cited in King's Citizen Petition. This is my opinion for several reasons.

1. Metaxalone has a long and safe history of use at the plasma concentration levels cited by King.

30. As noted above, metaxalone has had a long and safe history of use (over forty years) as an adjunct for the treatment of musculoskeletal conditions, with a recommended dosing of 800 mg three to four times daily. Doses as high as 4,000 and even 9,600 mg have also been shown safe and effective. *See* Morey, "Metaxalone, a New Skeletal Muscle Relaxant," *J. Am. Osteo. Ass'n*, 62:517-21(1963) (Ex. 3); Fathie, "A Second Look at a Skeletal Muscle Relaxant: a Double-Blind Study of Metaxalone," *Current Therapeutic Research*, Vol. 6., No. 11:677-83, 679 (1964) (Ex. 10); Fathie, "Musculoskeletal Disorders and Their Management with a New Relaxant," *Clinical Medicine* 72:678-83, 679, 682 (1965) (Ex. 4); Abrams, *Clinical Drug Therapy* 146 (1995) (Ex. 11); Carter, "A new muscle relaxant," Diseases of the Nervous System 1962; 23(2):98-100 (Ex. 5).

31. Such high dosing levels, and the fact that the drug is recommended as an adjunctive measure in combination with other therapies, indicate that metaxalone is a relatively

weak drug that is easily and well tolerated by the human body, and rapidly metabolized and eliminated. This conclusion is further supported by a 2002 Elan study entitled "A Study To Evaluate The Pharmacokinetics Of Skelaxin® (Metaxalone) 2x400 mg Tablet Administered To Young And Elderly Volunteers Under Fed And Fasted Conditions," in which the investigator concluded that "Skelaxin® was safe and well tolerated by the subjects." Ex. 25 at 34.

32. In addition, the literature reflects that metaxalone has been administered safely and effectively at the recommended dose of 800 mg three to four times daily both with and without food, and that this dosing schedule remained the same in both cases. *See* Ex. 3 at 518; Ex. 4 at 679, 682; Ex. 10 at 679, 683.

33. I also note that the drug was used safely and effectively for 40 years without any notification of any food-related bioavailability increase. *See* Harden, "A review of three commonly prescribed skeletal muscle relaxants," *J. of Back and Musculoskeletal Rehab.* 15:63-66 (2000) (stating with regard to metaxalone that "there are no reports in the literature of potentially dangerous side effects or safety concerns") (Ex. 9).

34. In my opinion, these facts demonstrate that the food-related bioavailability increase cited by King has no impact upon dosing of the drug or its safe or effective use.

2. The plasma concentration levels cited by King are clinically irrelevant to the safety or efficacy of metaxalone.

35. As also noted above, the mechanism of action, the precise site of action, the amount of metaxalone (or active metabolite) reaching the site of action, and the plasma concentrations required for its therapeutic and toxic effects are all unknown. Without this information, no relationship can be drawn between the safety and/or efficacy of metaxalone on the one hand, and the plasma concentration levels cited by King, on the other.

36. Furthermore, the current labeling states that various pharmacokinetic parameters increase in a "statistically significant[]"manner. However, King has made no attempt, either in the current labeling or in its Citizen Petition, to link statistical significance with clinical relevance. As the FDA has recognized, no such link necessarily exists. Ex. 9 to King's Citizen Petition at 11-12.

37. For these reasons, any differences between fed and fasted AUC(last), AUC(inf), and/or Cmax are not meaningful, do not correlate to any therapeutic effect, and have no clinical significance. Accordingly, there would be no justification to make dosing decisions based upon such information.

38. That the safety and efficacy of metaxalone are unrelated to known plasma concentration levels does not render metaxalone unique; the safety and efficacy of numerous other drugs similarly have no known correlation to their plasma concentration levels. Examples include proton pump inhibitors such as esomeprazole (Nexium) which has a half-life of about 3 hours, but inhibits stomach acid secretion in excess of 20 hours; the osteoporosis drug product alendronate sodium (Fosamax) prevents bone density loss long after it has reached undetectable levels in the blood plasma; and the hormone replacement therapy agent medroxyprogesterone acetate (Prempro) reaches undetectable levels in the blood plasma while still being effective against the symptoms of menopause. Such drugs are instead dosed in the quantities and on the schedules that have been proven safe and effective, as has long been the case with metaxalone.

3. Any difference between fed and fasted steady-state plasma concentration levels of metaxalone experienced with normal use of the drug would be insignificant or non-existent.

39. In my opinion, the information regarding fed-state bioavailability data cited by King is also not reflective of the normal use of metaxalone in several respects.

40. First, this fed-state data was generated from subjects who were administered metaxalone after the consumption of a high-fat meal. Such high-fat test meals are an unrealistic representation of the average American meal, and are instead used to create artificially increased fed-state bioavailability for test purposes only.

41. Additionally, each subject in the trials was given only a single 400 or 800 mg dose of metaxalone, whereas the recommended dose of metaxalone is 800 mg three to four times daily. Thus, the bioavailability data cited by King was generated by studies that did not follow the standard dosing regimen for metaxalone in terms of total daily milligrams or timing.

42. It is my opinion that when metaxalone is administered as recommended under normal eating conditions, any differences between fed and fasted bioavailability would be clinically insignificant. The reason for this, in part, is the half-life for a single 400 mg dose reported in King's labeling in the fasted state (9.2 hours) is longer than the half-life in the fed state (2.4 hours). Thus, fed patients will absorb, metabolize, and eliminate metaxalone more rapidly than fasted patients, thereby leaving the fasted patients with more residual metaxalone in the body at the time the next dose is administered. As this pattern is repeated during the recommended dosing regimen, the difference between fed and fasted plasma concentration levels would diminish to insignificant levels over time.

43. In addition, the average American meal would be expected to contain significantly less fat than the amount present in the test meal used in the bioavailability studies cited by King. When metaxalone is administered with a more realistic meal, the fed-state AUC(last), AUC(inf) and Cmax each will be significantly lower, thereby reducing the difference between fed-state and fasted-state AUC(last), AUC(inf) and Cmax to a negligible level.

44. Moreover, when administered q.i.d., the fourth daily dose of metaxalone – presumably administered at bedtime – would likely be administered with no meal at all, as

people do not typically consume meals, especially high-fat meals, immediately before bedtime. Such administration of a fourth daily dose of metaxalone without any meal would further reduce the daily average fed-state plasma concentrations of the drug.

45. Thus, in my opinion, when metaxalone is administered as recommended in the labeling (800 mg t.i.d. or q.i.d.) in conjunction with average meal consumption, any difference between fed and fasted AUC(last), AUC(inf) and Cmax would be expected to be small or non-existent.

4. The variability in the data cited by King further indicates that the cited plasma concentration levels are clinically irrelevant.

46. I also note that there exists a significant amount of variability in the bioavailability data cited by King, in terms of both half-life and Tmax, as compared to the bioavailability increases that King reports. For example, King's data reports the following variability in half-life and Tmax: 400 mg fasted half-life-9.2 hours +/-52%; 400 mg fed half life-2.4 hours +/- 50%; 400 mg fasted Tmax-3.3 hours +/- 36%; 400 mg fed Tmax-4.3 hours +/- 53%; 800 mg fasted half-life-8.0 hours +/- 58%; 800 mg fed half life-4.2 hours +/- 60%; 800 mg fasted Tmax-3.0 hours +/- 40%; 800 mg fed Tmax-4.9 hours +/- 47%.

47. This variability further demonstrates that the bioavailability of metaxalone frequently is not relatively increased by administration of the drug with food. As explained above, this is because absorption of the drug can often be higher in a fasted state if administration of the drug corresponds with the fasted-state secretion of bile into the duodenum every 90 minutes associated with Phase III of the MMC.

48. This variability is also demonstrated by the foregoing 2002 Elan study. Ex. 25. That study concluded that "[i]n the presence of food, the Cmax, AUC(last), and AUC(inf) were increased in both age groups, although these differences did not reach statistical significance."

Ex. 25 at 34. This study further demonstrates the variability and uncertainty of a food-induced increase in bioavailability even when a high-fat meal is administered.

49. I also note that the recommended dosing of metaxalone has a long-accepted variation of 33% as patients can be dosed 800 mg t.i.d. (2400 mg) or q.i.d. (3200 mg). In addition, as noted previously, the literature reflects that substantially higher doses of 4,000 and even 9,600 mg have been demonstrated safe and effective. These reported daily dosing levels, compared with a dosing of 800 mg t.i.d., would induce a variation of 67% and 300%, respectively.

50. In view of the variability in King's data, the long-accepted 33% variability in the recommended dosing of metaxalone (between 2,400-3,200 mg daily), and the significantly higher doses (9,600 mg) that have been demonstrated safe and effective, the relative fed-state bioavailability increases cited by King (15.4% for AUC(inf), 23.5% for AUC(last) and 77.5% for Cmax for the administration of a single 400 mg dose, and 94% AUC(inf) and 42% Cmax for the administration of 800 mg) are clearly not clinically relevant to the safe or effective use of the drug.

5. The food-effect data cited by King is clinically irrelevant to the determination of any potentially new drug-drug interactions between metaxalone and another drug product.

51. In my opinion, the food-effect data cited by King is also clinically irrelevant to the determination of any potential pharmacokinetic drug-drug interactions that have not yet been identified, which I believe would be unlikely, in any event. I also note that known therapeutic interactions are stated on the package insert.

52. The fundamental requirements for determining a potential pharmacokinetic drug-drug interaction (as opposed to therapeutic interactions which are already identified in the labeling) between metaxalone (as the substrate) and an interfering drug are an understanding of

how metaxalone is metabolized by the enzymes in the gut mucosa, knowledge of the enzymes responsible for hepatic metabolism and the resulting metabolites as well as the route of eventual elimination. As pointed out by Dr. Benet in his declaration, none of this information is known for metaxalone. *See* Ex. 10 to King's Citizen Petition at ¶ 27 (stating that at the present time there is "no information as to the metabolic profile of metaxalone").

53. Thus, because the metabolic profile for metaxalone is unknown, meaningful study of potential pharmacokinetic drug-drug interactions is not possible at the present time, as there is no way to select classes of drugs that may potentially interact with metaxalone. This point is recognized in the FDA's Guidance cited by King, which states that:

> In testing an investigational drug for the possibility that its metabolism is inhibited or induced (i.e., as a substrate), selection of the interacting drugs should be based on in vitro or other metabolism studies identifying the enzyme systems that metabolize the drug. **The choice of interacting drug should then be based on known, important inhibitors of the pathway under investigation.**

Ex. 9 to King's Citizen Petition at 9 (emphasis added).

54. In addition, as explained above, metaxalone has been used primarily as an adjunct in combination with other therapies, and it has a wide "therapeutic index," in that safe and effective daily dosing has been reported to be between 2400 and 9600 mg. In view of metaxalone's common use and its wide therapeutic index, it is not likely to have any unknown drug-drug interactions after 42 years of use. Indeed, drug-drug interactions are "most obvious and expected for a drug with a narrow therapeutic range." Ex. 9 to King's Citizen Petition at 3.

55. Additionally, the effect on fed-state bioavailability of metaxalone cited by King would be unrelated to the determination of any possible unknown drug-drug or therapeutic interactions. As noted above, drug-drug interactions are discovered through analysis of a drug's metabolic profile, not by analyzing plasma concentration versus time curves.

56. Moreover, to the extent that drug-drug interactions with metaxalone exist, they are already addressed in the current labeling with the information located under the heading "Drug Interaction." It is my understanding that Corepharma does not seek, nor has the FDA allowed, a carve-out of any part of the "Drug Interaction" portion of the labeling.

57. In my opinion, the drug-drug interactions identified by Dr. Benet in his declaration bear no relationship to metaxalone and are irrelevant to the issues raised in King's Citizen Petition. Ex. 10 to King's Citizen Petition at ¶ 28; *see also* King Citizen Petition at 15. None of the drug-drug interactions identified by Dr. Benet are skeletal muscle relaxants; none are structurally related to metaxalone; and none (except digoxin) have been used in mass quantities for over 40 years.

58. The first drug-drug interaction mentioned by Dr. Benet is the well-publicized diet drug combination phen-fen. This example is inapplicable to metaxalone as both phentermine and fenfluramine were individually approved for the treatment of obesity. However, the heart-valve disease associated with the use of these two drugs was a result of an unapproved, off-label, combination of the two drugs. Fenfluramine and phentermine were never approved by the FDA for use together. To my knowledge, a similar situation for metaxalone has not been reported.

59. The second drug mentioned by Dr. Benet is terfenadine. Terfenadine was first approved in 1985 and the cardiac problems associated with the co-administration of terfenadine with either ketoconzole or certain macrolide antibiotics became known shortly after approval. By 1992 the FDA had revised the terfenadine labeling to include a prominent boxed warning cautioning against its use with certain drugs that inhibit the metabolism of terfenadine. Terfenadine was only available for a very brief time before drug-drug interactions became known and the FDA mandated labeling changes. Thus, the terfenadine situation is far different from the 40 years of safe use of metaxalone with no reported drug-drug interactions.

60. Another drug-drug interaction mentioned by Dr. Benet is quinidine and digoxin. Although digoxin has been used for over 60 years, it has been used only in a specific cardiac patient group by cardiologists. In addition, the interaction between quinidine and digoxin is inapposite to any unknown drug interactions with metaxalone. As previously stated, metaxalone has a very broad therapeutic index as the recommended daily dose varies by 33% (800 mg t.i.d. or q.i.d.) and the literature has reported other doses as high at 9,600 mg without any reports of toxicity. Furthermore, the approved Skelaxin labeling states that animal studies failed to yield an LD_{50} value in dogs and the literature reports a large LD_{50} of 1200 mg/kg in rats. Ex. 13 at 284. In stark contrast, digoxin is administered on a scale of 0.1 mg/dose (8000 times smaller than each recommended dose of metaxalone) and has an extremely narrow therapeutic index with safe and effective blood concentration levels reported to be between .9 ng/ml and 2.0 ng/ml, which is approximately 400 to 800 times less than the fasted plasma concentration levels reported in the current labeling for Skelaxin. In my opinion, similar arguments for the therapeutic use of quinidine would place it closer to digoxin than to metaxalone.

61. The final drug-drug interaction mentioned by Dr. Benet is fexofenadine and ketoconazole. The interaction between fexofenadine and ketoconazole is similarly irrelevant to any possible drug interactions with metaxalone. First, ketoconazole is a well-known and strong inhibitor of the CYP3A4 hepatic enzyme and known to interfere with the metabolism of numerous drugs, including fexofenadine. Second, the fexofenadine/ketoconazole drug-drug interaction warning was added to the fexofenadine labeling in 1998 – just two years after approval of fexofenadine. Thus, the drug-drug interaction determination and warning occurring very soon after approval of fexofenadine is significantly different from metaxalone which has been safely used without known interactions for over 40 years.

D. The Effects of Age and Gender on the Bioavailability of Metaxalone are Clinically Irrelevant

62. In my opinion, the age and gender effects cited in King's Citizen Petition (to the extent those effects do occur), are irrelevant.

63. According to King's Citizen Petition and the declarations of Drs. Elia and Benet, "there is a gender effect in that bioavailability of the drug is higher in females than in males, and an age effect in that bioavailability of the drug increases with the age of the patient." King Citizen Petition at 16; *see also,* Elia Declaration at ¶¶ 21-26; Benet Declaration at ¶¶ 22-24. I note that while King and its experts describe this effect as "statistically significant," neither King nor its experts recommends a food instruction or dosing adjustment to minimize it. That alone indicates that this gender effect (to the extent it actually occurs) is irrelevant. In my opinion, the gender and age effect would be irrelevant for the same reasons that the fed-state bioavailability food effect is clinically irrelevant as explained above.

64. Similarly, according to King, there is an age-effect with respect to metaxalone "in that bioavailability of the drug increases with the age of the patient" only when the drug is administered in a fasted state. King's Citizen Petition at 16. I also understand from King's Citizen Petition that this age-effect (as well as the foregoing gender- effect) is based upon a "meta-analysis" of four studies, which I note are discussed in United States Patent Application No. 10/420,804 (the '804 patent application), dated April 23, 2003. Ex. 21. In analyzing two of the four studies, the '804 patent application concluded as follows:

> [T]he magnitudes of the estimated effects of age under fasted conditions can be more easily illustrated by considering the percentage increases when comparing a 70-year-old individual to a 20-year-old individual. On the natural log scale, these increases are 10%, 7%, and 7% for ln(Cmax), ln(AUC(last)), and ln(AUC(inf)), respectively.

Ex. 21 at ¶ 69.

65. It is my opinion that these increases in pharmacokinetic parameters are clinically and statistically insignificant. First, the magnitude of the change over a 50 year period is very small, especially when their magnitude are compared to the very wide therapeutic index for metaxalone. Additionally, changes on the order of 7-10% are well within the range of 80% to 125% (-20% to +25%) that are regarded as a clinically insignificant variation and thus bioequivalent. Ex. 8 to King's Citizen Petition at 7.

66. Furthermore, the 7-10% variation in pharmacokinetic parameters over 50 years due to age is insignificant when compared to the variability in reported metaxalone half-life (fasted 9.2 hours +/- 52%; fed 2.4 hours +/- 50%) with the administration of a single 400 mg tablet and the reported variability in half-life (fasted 8.0 hours +/-58%; fed 4.2 hours +/-60%) with the administration of two 400 mg tablets.

67. Finally, the 7-10% variation in the bioavailability of metaxalone due to age is negligible as it is well within the expected variation of a randomly selected patient population, thus having little, if any, clinical relevance.

68. It is also my opinion that the age-effect described by King is not supported by the data submitted with its Citizen Petition and is not reliably based on the studies that King describes for several reasons.

69. First, I note that King's Citizen Petition includes no per-patient raw data. Metaxalone blood plasma concentration levels for each blood draw from each patient should be provided in order to analyze and substantiate the statistical analysis underpinning the conclusions reached by King and its experts.

70. In addition, assuming the raw data supports King's statistical analysis, the conclusions regarding the effect of age on the bioavailability of metaxalone are still unjustified,

in my opinion, because they were based upon pooling the results of only two studies to yield a total population size of 103 subjects. Ex. 21 at ¶¶ 54-55.

71. In addition, a population size of 103 is much too small to draw any reliable conclusions regarding such a small statistical effect. Typical population sizes used in the pharmaceutical industry to conduct correlation analyses are usually 10 to 50 times larger than the combined size of the two studies relied upon by King.

72. Finally, in order to reach the conclusion that there is any age effect on fasted bioavailability at all, King was required to pool the populations in the two studies together in order to identify even the small statistical effect cited. Ex. 21 at ¶¶ 54-55. This pooling or "meta-analysis" of multiple studies detracts from the already marginal influence that age has on fasted bioavailability of metaxalone.

73. My conclusion that the variation in bioavailability of metaxalone due to age is clinically irrelevant is further supported by the information set forth in the '804 patent application. The linear regression coefficient of determination (R^2) set forth in the '804 patent application for the ln-transformed age effect data is only .282, .264 and .260 for Cmax, AUC(last) and AUC(inf), respectively. Ex. 21 at ¶ 67. An R^2 value of 1.000 would mean that all of the variation in a fasted pharmacokinetic parameter would be due to age while an R^2 value of 0.000 would mean that none of the variation in fasted Cmax, AUC(last) and AUC(inf) would be due to age. Thus, 71.8% (1.000 - 0.282) of the variation in Cmax (which is only 10% between a 20 year old and a 70 year old) is due to a factor other than age; 73.6% of the variation in AUC(last) (which is only 7% between a 20 year old and a 70 year old) is due to a factor other than age; and 74.0% of the variation in AUC(inf) (which is only 7% between a 20 year old and a 70 year old) is due to a factor other than age.

74. The presence of very low values of R^2 when attempting to show a relationship between fasted-state bioavailability and age demonstrates that there is very little correlation between age and fasted Cmax, AUC(last) and AUC(inf). Due to the very low correlation between age and fasted pharmacokinetic parameters, the 7-10% increase in Cmax, AUC(last) and AUC(inf) is clinically irrelevant.

75. The clinical insignificance of a 7-10% increase in ln-transformed Cmax, AUC(last) and AUC(inf) is further supported by comparing this minimal age-effect variation in the fasted state with the long-accepted variation in the recommended dosing of 33% (800 mg t.i.d. or q.i.d.). This comparison manifestly demonstrates that a 7-10% variation in Cmax, AUC(last) and AUC(inf) is insignificant in comparison to the accepted dosing variation that has been recommended in the label, described in the literature (Ex. 3 at 518; Ex. 4 at 679, 682; Ex. 10 at 679, 683) and used since 1962.

IV. CONCLUSION

In sum, the fed-state bioavailability data should be permitted to be carved out of the generic labeling for metaxalone. Based upon my nearly 50 years of experience in pharmacology, it is my opinion that the carved-out fed-state bioavailability data is clinically irrelevant in terms of both its magnitude and how it was generated. Moreover, the 42 years that metaxalone has been used without any fed-state bioavailability information manifestly demonstrates that it is a safe and effective drug without this information in the drug's labeling.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge, information, and belief.

April 28, 2004
Date

Paul Bass, Ph.D.

CURRICULUM VITAE

Paul Bass
Professor of Pharmacology
Center for Health Sciences
University of Wisconsin
Madison, Wisconsin
53706

Appendix A

CURRICULUM VITAE

Paul Bass

Place of Birth: Winnipeg, Canada

Citizenship: U.S.

Office: (608) 262-5753, Fax (608) 262-3397
email: pbass@pharmacy.wisc.edu

Home: (608) 238-4184, Fax (608) 238-8033
email: pbass@facstaff.wisc.edu

Education:

Undergraduate

University of British Columbia, Vancouver, British Columbia B.S.P. (Pharmacy), 1953

Graduate

University of British Columbia
M.A. (Pharmacology), 1955
Major advisor: Dr. E. E. Daniel

Thesis: Changes in the function and ionic composition of the alimentary tract in response to dietary cation deficiencies, and the possible role of adrenal medullary and cortical hormones in mediating these responses.

McGill University, Montreal, Quebec
Ph.D. (Pharmacology), 1957
Major advisor: Dr. K. I. Melville

Thesis: The role and possible significance of potassium, calcium, magnesium in the cardiac actions of digitalis glycosides.

Postdoctorate

Postdoctoral student for 12 months in the Departments of Physiology and Biochemistry, McGill University, 1957-1958.

Fellow for 18 months in the gastrointestinal section of the Department of Physiology, Mayo Foundation, 1958-1960.

Positions Held:

London Drugs
Vancouver, British Columbia
Title: Manager, 1953-1954.

Ayerst, McKenna and Harrison
Montreal, Quebec
Title: Research Assistant, June-August, 1956.

Parke, Davis and Company
Detroit, Michigan
Position accepted with Parke Davis and Company, April 1, 1957. The postdoctoral training periods were sponsored by the Company. Initiated research with Parke, Davis at Ann Arbor, January 1960.

Title: Research Pharmacologist, 1960-1966
Senior Research Pharmacologist, 1967-1968
Associate Laboratory Director, 1968-1970.

Academic Appointment:
Concomitant with Employment at Parke Davis:
Lecturer, Department of Pharmacology, University of Michigan
1966 to 1970.

University of Wisconsin
Madison, Wisconsin
Title: Dual appointment 1970 to present
(a) Professor, School of Pharmacy (50% effective 2/98)
(b) Professor of Pharmacology, School of Medicine

and Professor of Pharmacology, School of Veternary Medicine (1980-84)

Society Memberships:

American Society for Pharmacology and Experimental Therapeutics
American Gastroenterological Association
Society for Experimental Biology and Medicine
Pharmacological Society of Canada
Sigma XI
Alumni Association of Mayo Foundation

Member of Editorial Board:

American Journal of Physiology 1976-79, 1982-1991
Journal of Pharmacology and Experimental Therapeutics 1980-1999
Journal of Pharmacological Methods, 1983-98
Neurogastroenterology and Motility, 1988-98

Review Publications for the following:

Journal of Pharmacology and Experimental Therapeutics
Gastroenterology
American Journal of Physiology
American Journal of Digestive Diseases
Journal of Medicinal Chemistry

Invited Participant by the following groups:

1. Gastroenterology Research Group, American Gastroenterology Association, 1964 (Chicago). Symposium on Gastrointestinal Smooth Muscle.

2. Gordon Research Conference, 1968 (Crystal Mountain, Washington). Symposium on Toxicology and Safety Evaluation.

3. Gastrointestinal Pharmacology Group, American Physiological Society, 1968 (New York). Workshop on Methodology in Gastrointestinal Motility and Gastric Secretion.

4. East-West Conference, National Science Foundation, 1968 (Hawaii). Conference on Clinical Application of Smooth Muscle Electromyography.

5. University of Erlangen, Dept. of Medicine, 1969 (Nurnberg, Germany). International Symposium on Motility of the G. I. Tract.

6. American Chemical Society, 1969 (New York). Chairman of Symposium on Agents Affecting Gastrointestinal Function.

7. American Journal of Digestive Diseases, Guest Editor: Symposium on Agents Affecting Gastrointestinal Function. Vol. 15, 1970.

8. International Motility Symposium, 1971 Saltzjobaden, Sweden; 1973 Banff, Canada; 1975 Leuven, Belgium; 1977 Edinburgh, Scotland; 1979 Iowa City, U.S.A.; 1981 Koeningstein, Germany; 1983 Aix en Provence, France; 1985 Rochester, U.S.A.; 1989 Gmunden, Austria; 1991 Kobe, Japan.

9. MUCIA (Midwest Universities Consortium for International Activities) Advisor and visiting professor to the Department of Physiology and Pharmacology, Faculty of Veterinary Medicine, Bogor Agricultural University and University of Gudjah Mudah, Jug Jakarta, Indonesia (June 1-30, 1974).

10. Algerian Medical School, Pharmacologist with committee of 10 from the University of Wisconsin, sponsored by the U.S. State Department, to assess Algeria's medical needs. May 1 to 10, 1977.

11. Department of Pharmacology, Hebrew University, Jerusalem, Israel. Visiting professor, 1 week, September 1977.

12. East-West Conference, National Science Foundation, 1977 (Hawaii). Conference on Smooth Muscle Electromyography.

13. National Institute of Health, Member of Special Review Committee of Site Visit Panel, of Peptic Ulcer Center, VA Wadsworth Hospital Center, Los Angeles, February 1978.

14. APhA Academy of Pharmaceutical Sciences, 1978, Symposium on Gastrointestinal Absorption of Drugs.

15. NIH, 1979, Workshop on Irritable Bowel Disease, Bethesda, MD.

16. Continuing Education, School of Pharmacy, Rutgers, 2 Lectures, 1979.

17. Warner Lambert, Ann Arbor, 1980, Symposium on Drugs of the 1990's.

18. Symposium, American Motility Society, 1982, Brookline, Mass. Clinical Disorders of Esophagus and Gastric Emptying.

19. Symposium on Functional Disorders of the Digestive Tract, 1982, Rochester, NY.

20. NATO Advanced Study Institute, 1983 (Erice, Italy) Gastrointestinal Tract Drugs.

21. U.S.-Japan Seminar, National Science Foundation, 1985 (Hawaii) on Gastrointestinal Smooth Muscle in Health and Disease.

22. Dupont-Alza Workshop, 1985 (Nassau, Bahamas), on Therapeutic Prospects for Colon-Targeted Drug Delivery Systems.

23. Am. Gastroenterological Assoc., 1985 (New York) Organized and Chaired Symposium on drugs and inflammatory bowel disease.

24. Merrell-Dow workshop, 1986 (Phoenix) organized and chaired symposium on fiber in the American diet.

25. American Motility Society, 1986 (Houston) Chaired Nominating Committee for Society.

26. Vicks Research Symposium, 1986 (Sheldon, CT) on aromatics and pulmonary function.

27. General Medicine Study Section, 1987, 1988 National Institute of Health, DHH, February meetings to review grants.

28. Morris Animal Foundation, 1987 (Indianapolis), 1990 (Chicago) Panel on Bloat in the Dog.

29. American Gastroenterological Association, 1987 (Chicago), Inflammatory Bowel Disease Forum on Drugs.

30. Jouveinal Laboratories, 1989, Workshop on Stress and Digestive Motility, Mont Gabriel Canada.

31. Procter and Gamble Co, (February) 1990, Cincinnati, Chaired External Advisory Panel Review of G.I. Motility Disorders.

32. Procter and Gamble Co., September 1990, Egam, England, Member of International Workshop on Methods for Antitussive Studies.

33. Second Jerusalem Conf. on Pharm. Sci. and Clin. Pharm., May 1992 (Jerusalem), organized and chaired session on G.I. parameters that influence oral medication.

34. Capsugel, Warner-Lambert, April 1993 (Short Hills, N.J.), Symposium on Targeted Drug Delivery to the G.I. Tract.

35. American Association of Pharmaceutical Science, November 1993 (Orlando, FL), organized and chaired symposium on colonic drug delivery.

36. NIH, December 1993 (Herndon, VA), workshop on oral drug delivery: Interface between discovery and development.

The University of Wisconsin-Madison committees:

Member or chairman of numerous committees that govern policies for the entire campus as well as School of Pharmacy, Medical School, Center for Health Science, School of Veterinary Medicine.

Offices in other professional activities:

Member of Steering Committee and Chairman 1968-69 of the Gastrointestinal Pharmacology Group.

Member of the Steering Committee 1970-73, Chairman 1972-73, Gullet Club affiliate of the American Gastroenterological Association.

Co-chairman of Finance Committee of the International Motility Symposium 1973.

Member of Human Research Advisory Committee Warf Institute Inc., Madison, 1975-78.

Consultant to the Department of Surgery, Veterans Administration Hospital, Milwaukee, 1974-78

Member of Drug Quality Council, State of Wisconsin, by Governor Appointment, 1980-1984

Member, United States Pharmacopea Drug Information Advisory Panel; Gastroenterology and Nutrition, 1981-1985, 1990-

Member, Advisory Board of the Inflammatory Bowel Disease Forum, affiliate of the Am. Gastroenterological Assoc., 1983-

Founder and Current Chair of Midwest Motility Society, 1985-

Founder & Member of Executive Committee of Section of Gastrointestinal Pharmacology, American Society for Pharmacology and Experimental Therapeutics, 1992-.

Founder & Member of Gastrointestinal Pharmacology, International Union of Pharmacology, 1993-.

Member, Scientific Advisory Board, Clarion Pharmaceutics Inc., 1993-.

Grants and Awards, 1970-

NIH (Direct Costs Only)	1971-74	Contractions and emptying of stomach and intestine	98,808
		Laxative effects on dog intestinal motility	14,046
	1975-78	Flow and small bowel contractions	138,000
	1975-79	Predoctoral Pharmacological, Toxicological Science (Training Grant)	267,225
	1976-80	Motor, electric properties of stomach and small bowel	134,774
	1983-86	Enteric neuron ablation: altered motility and section	99,189
	1986-91	Enteric neuron ablation: altered motility and secretion	428,030
	1994-95	Distributions of cryptospiridium antibody in the rat (R43)	17,500
	1998-2001	Regulation of intestinal function by adult tapeworm (RO1, Co-Investigator)	477,679
U.S. Israel Bi-national Sci. (collab. with A. Rubinstein)	1994-97	Mucosal attachment of cationic antioxidant enzymes	100,000
Surgical Associates Wisconsin	1971-72	Effects of parietal cell and truncal vagotomy on gastric emptying	6,500
	1972-73	Effects of parietal cell and truncal vagotomy on gastric emptying	6,500
Graduate School University of Wisconsin	1970-72	Basic studies in gastroenterology	(per year) 5,000
	1979-80	Basic studies in gastroenterology	5,000
	1981-82	Basic studies in gastroenterology	7,500
	1987-88	Development of a 24-hour esophogeal pressure recorder	12,182
	1993-94	Enteric neuron ablation	16,000
Allocation from	1974-75	Effect of laxatives on intestinal motor	3,000

GRS Funds		patterns	
	1976-77	Research on laxatives	1,800
	1980-81	Research on dietary fiber	8,000
Pharmaceutical Companies	1972-74	Research on gastrointestinal motility	5,500
	1979-80	Research on gastrointestinal motility	5,000
	1981-82	Research on gastrointestinal motility	25,000
	1983-84	Research on gastrointestinal motility	28,000
	1990-91	Research on epidermal growth factor	53,000
	1991-92	Research on dietary fiber	20,000
	1993-94	Research on dietary fiber	30,000
	1994-95	Use of sucralfate as a radioprotectant	50,000
	1997-	Antitussive Study Proctor & Gamble Gift	55,000 15,000

Specific Teaching and Instructional Activities:

Pharmacology and Toxicology - Undergraduate UW-Madison	60 hrs. per year
Graduate Program - Seminars Research Supervision	20 hrs. per week
Gastroenterology - School of Medicine	15 hrs. per year
Continuing Education	10 hrs. per year

Pharmacology - Medical College of Wisconsin, Milwaukee (1972-74)

Major Research Professor for: A. completed doctorate degree

University of Michigan
Henry Jacoby, 1964
Gerald Carlson, 1969
Norman Weisbrodt, 1970

University of Wisconsin
Timothy Gaginella, 1974
Herbert Ormsbee, III, 1974
John Stewart, 1975
Gary Gullickson, 1979
James Russell, 1984
Deborah A. Fox, 1985
James R. Herman, 1988

Norman A. See, 1991
Michael S. Luck, 1994
Mark A. Osinski, 1994

B. completed master degree

Everett Engstrom, 1976
Mark Sender, 1977
Bruce Teeter, 1978
James Russell, 1980
Deborah Fox, 1983
Norman A. See, 1987
Bader E. Bellahsène, 1988
Catherine J. Pfister, 1989

Publications: This consists of over 120 research publications, 27 chapters, and reviews and 87 abstracts.

Representative papers from the last few years are:

1. Evaluation of Early Events in the Creation of Amyenteric Opossum Model of Achalasia. C. Singaram, M. A. Sweet, E. A. Gaumnitz, P. Bass, and R. L. Snipes. Neurogastroenterol. Mot. 8, 351-361 (1996).

2. Tapeworm Infection Decreases Intestinal Transit and Enteric Aerobic Bacterial Populations. M. B. Dwinell, P. Bass, D. M. Schaefer, and J. A. Oaks. Am. J. Physiol. 273, G480-G485, 1997.

3. *Hymenloepsis diminuta*: Mucosal Mastocytosis and Intestinal Smooth Muscle Hypertrophy Occur in Tapeworm-Infected Rats. M. B. Dwinell, R. M. Wise, P. Bass, and J. A. Oaks. Exp. Parasitol. 89, 92-102 (1998).

4. *Hymenloepsis diminuta* Fractions But Not Previous Tapeworm Infection Stimulate Intestinal Myoelectric Alterations In Vivo in the Rat. M. B. Dwinell, P. Bass, and J. A. Oaks. J. Parasitol. 84(4), 673-680 (1998).

5. Peripheral and Central Actions of Orphanin FQ (Nociceptin) on Murine Colon. M. A. Osinski, P. Bass, and E. A. Gaumnitz. Am. J. Physiol. 276, G125-131 (1999).

6. Differences in the Reducing Power Along the Rat GI Tract: Lower Antioxidant Capacity of the Colon. S. Blau, A. Rubinstein, P. Bass, C. Singaram, and R. Kohen. Mol. and Cell. Biochem. 194, 185-191 (1999).

7. Relation Between Colonic Inflammation Severity and Total Low-Molecular-Weight Antioxidant Profiles in Experimental Colitis. S. Blau, R. Kohen, P. Bass and A. Rubinstein. Dig. Dis. & Sci. 45(6), 1180-1187 (2000).

8. The Effect of Local Attachment of Cationized Antioxidant Enzymes on Experimental Colitis in the Rat. S. Blau, R. Kohen, P. Bass, and A. Rubinstein. Pharmaceutical Research 17(9), 1077-1084 (2000).

Paul Bass

RESEARCH PUBLICATIONS (excluding publications on preceding page)

1. Responses of smooth and striate muscle to alterations in extracellular electrolytes. E. E. Daniel and P. Bass, Am. J. Physiol., 187, 247-252 (1956).

2. Influence of sodium, potassium and adrenal hormones on gastrointestinal motility. E. E. Daniel and P. Bass, Am. J. Physiol., 187, 253-258 (1956).

3. Effects of magnesium on coronary flow and heart action and its influence on the responses to adrenaline and noradrenaline. P. Bass, I. Mazurkiewicz, and K. I. Melville, Arch. Int. Pharmacodyn., 117, 9-22 (1958).

4. Absorption of water, sodium and potassium in small intestine of dogs. C. F. Code, P. Bass, G. B. McClary, Jr., R. L. Newnum, and A. L. Orvis, Am. J. Physiol., 199, 281-288 (1960).

5. Motor and electric activity of the duodenum. P. Bass, C. F. Code, and E. H. Lambert, Am. J. Physiol., 201, 287-291 (1961).

6. Electrical activity of gastroduodenal junction. P. Bass, C. F. Code, and E. H. Lambert, Am. J. Physiol., 201, 587-592 (1961).

7. In vivo extraluminal contractile force transducer for gastrointestinal muscle. H. I. Jacoby, P. Bass, and D. R. Bennett, J. App. Physiol., 18, 658-665 (1963).

8. Chronic electrical activity of gastroduodenal area: effects of food and certain catecholamines. E. J. McCoy and P. Bass, Am. J. Physiol., 205, 439-445 (1963).

9. Certain pharmacological properties of 3-ethyl-2-methyl-2-phenylsuccinimide. G. Chen and P. Bass, Arch. Int. Pharmacodyn., 152, 115-120 (1964),.

10. Simultaneous recording of electrical and mechanical activity of the uterus in the unanesthetized animal. P. Bass and M. R. Callantine, Nature, 203, 1367-1368 (1964).

11. Electrical and extraluminal contractile-force activity of the duodenum of the dog. P. Bass and J. N. Wiley, Am. J. Dig. Dis., 10, 183-200 (1965).

12. Effects of ligation and morphine on electric and motor activity of the duodenum of dog. P. Bass and J. N. Wiley, Am. J. Physiol., 208, 908-913 (1965).

13. Synthesis and pharmacological action of 3-amino-2, 1-benzisothiazoles. R. F. Meyer, B. L. Cummings, P. Bass, and H. O. J. Collier, J. Med. Chem., 8, 515-519 (1965).

14. Prolonged administration of atropine or histamine in a silicone rubber implant. P. Bass, R. A. Purdon, and J. N. Wiley, Nature, 208, 591-592 (1965).

15. Gastric antisecretory and other pharmacologic studies on 2,2'-bipyridine. P. Bass, R. A. Purdon, M. A. Patterson, and D. E. Butler, J. Pharmacol. and Exp. Ther., 152, 104-115 (1966).

16. Gastric antisecretory and other pharmacological studies of 3-methylamino-2,1-benzisothiazole. P. Bass, R. A. Purdon, and M. A. Patterson, J. Pharmacol. and Exp. Ther., 153, 292-300 (1966).

17. Gastric secretory responses to drugs affecting adrenergic mechanisms in rats. P. Bass and M. A. Patterson, J. Pharmacol. and Exp. Ther., 155, 142-149 (1967).

18. Contractile and electric activity of the extrahepatic biliary tract and duodenum. J. R. Ludwick and P. Bass, Surg. Gynec. and Obstet., 124, 536-546 (1967).

19. Effect of chelators and divalent cations on gastric secretion in the rat. P. Bass, D. E. Butler, M. A. Patterson, and R. A. Purdon, Arch. Int. Pharmacodyn., 169, 131-138 (1967).

20. Extraluminal contractile-force and electrical activity of reversed canine duodenum. J. R. Ludwick, J. N. Wiley, and P. Bass, Gastroenterology, 54, 41-51 (1968).

21. Differential response of body and antrum of canine stomach to various stimulants. J. J. Anderson, R. J. Bolt, B. M. Ullman, and P. Bass, Am. J. Dig. Dis., 13, 147-156 (1968).

22. The effect of bile and fat on gastric motility under the influence of various stimulants. J. J. Anderson, R. J. Bolt, B. M. Ullman, and P. Bass, Am. J. Dig. Dis., 13, 157-167 (1968).

23. A relation between gastroduodenal muscle contractions and gastric emptying. N. W. Weisbrodt, B. F. Overholt, J. N. Wiley, and P. Bass, Gut, 10, 543-548 (1969).

24. Pyloroplasty and Vagotomy: Early effects on antral and duodenal contractile activity. J. R. Ludwick, J. N. Wiley, and P. Bass, Arch. Surg., 99, 553-559 (1969).

25. Separation of the effect of alpha and beta adrenergic receptor stimulation on taenia coli. N. W. Weisbrodt, C. C. Hug, Jr., and P. Bass, J. Pharmacol. and Exp. Ther., 170, 272-280 (1969).

26. Gastric emptying following Finney pyloroplasty and vagotomy. J. R. Ludwick, J. N. Wiley and P. Bass, Am. J. Dig. Dis., 15, 347-352 (1970).

27. Effects of nicotine on gastric antral and duodenal contractile activity in the dog. G. M. Carlson, R. W. Ruddon, C. C. Hug, Jr., and P. Bass, J. Pharmacol. and Exp. Ther., 172, 367-376 (1970).

28. Analysis of the site of nicotine action on gastric antral and duodenal contractile activity. G. M. Carlson, R. W. Ruddon, C. C. Hug, Jr., S. K. Schmieg, and P. Bass, J. Pharmacol. and Exp. Ther., 172, 377-383 (1970).

29. Relationship of the enterohepatic cycle to ulcerogenesis in the rat small bowel with flufenamic acid. J. Wax, W. A. Clinger, P. Varner, P. Bass, and C. V. Winder, Gastroenterology, 58, 772-780 (1970).

30. Effect of nicotine and tyramine on contractile activity of the colon. N. W. Weisbrodt, C. C. Hug, Jr., S. K. Schmiege and P. Bass, Europ. J. Pharmacol., 12, 310-319 (1970).

31. Contractile activity of gastric antrum and taenia coli in the unanesthetized monkey. N. W. Weisbrodt, C. C. Hug, Jr., J. N. Wiley and P. Bass, J. App. Physiol., 30, 276-280 (1971).

32. Novel pharmacological activity of a series of substituted pyridines. D. E. Butler, P. Bass, I. C. Nordin, F. P. Hauck, Jr., and Y. J. L'Italien, J. Med. Chem., 14, 575-579 (1971).

33. Adrenergic mechanisms in the relation of guinea-pig taenia coli in vitro. L. M. Weisenthal, C. C. Hug, Jr., N. W. Weisbrodt, and P. Bass, J. Pharmacol. and Exp. Ther., 178, 497-508 (1971).

34. Effects of catecholamines on acid secretion by bullfrog isolated gastric mucosa. C. D. Thorpe, R. A. Frusco, P. Bass, and C. C. Hug, Jr., Surgical Forum, 22, 317-319 (1971).

35. The effect of secretin on the fed pattern of gastric and duodenal contractile activity. G. D. Walker, J. J. Stewart, and P. Bass, Surg. Gyn and Obst., 134, 807-809 (1972).

36. Contractile force transducer for recording muscle activity in unanesthetized animals. P. Bass and J. N. Wiley, J. App. Physiol., 32, 567-570 (1972).

37. Measurement of fecal output in rats. P. Bass, J. A. Kennedy and J. N. Wiley, Am. J. Dig. Dis., 17, 925-928 (1972).

38. The absorption and distribution of isopropamide iodide in the rat. T. S. Gaginella, P. Bass, J. H. Perrin and J. J. Vallner, J. Pharm. Pharmac., 25, 270-271 (1973).

39. Gastric and intestinal transit in rats measured by a radioactive test meal. R. A. Purdon and P. Bass, Gastroenterology, 64, 968-976 (1973).

40. Synthetic antidiarrheal agents. 1. an approach to the separation of antidiarrheal activity from narcotic analgesic activity. D. E. Butler, R. F. Meyers, S. M. Alexander, P. Bass and J. A. Kennedy., J. Med. Chem., 16, 49-54 (1973).

41. Effect of bile salts on partitioning behavior and GI absorption of a quaternary ammonium compound, isopropamide iodide. T. S. Gaginella, P. Bass, J. H. Perrin and J. J. Vallner, J. Pharm. Sci., 62, 1121-1125 (1973).

42. CI-750, a novel antidiarrheal agent. P. Bass, J. A. Kennedy, J. N. Wiley, J. Villarreal and D. E. Butler, J. Pharmacol. Exp. Ther., 186, 283-298 (1973).

43. Nicotine: Release from silicone rubber implants in vivo. T. S. Gaginella and P. Bass, Res. Commun. Chem. Pathol. Pharmacol., 7, 213-216 (1974).

44. Parietal cell vagotomy and gastric emptying of liquids in the dog. C. O. Weddle II, A. C. Springfield, H. S. Ormsbee III, and P. Bass, Arch. Surg., 108, 83-86 (1974).

45. The effects of liquid test meals and drugs on canine antralduodenal contractile activity following parietal cell or truncal vagotomy with or without pyloroplasty. H. S. Ormsbee III, A. C. Springfield, C. O. Weddle II, and P. Bass, Proc. Fourth International Symposium on Gastrointestinal Motility, Mitchell Press, 555-569 (1974).

46. The effect of parietal cell and truncal vagotomy on gastric and duodenal contractile activity of the unanesthetized dog. G. D. Walker, J. J. Stewart, and P. Bass, Ann. of Surg., 179, 853-585 (1974).

47. Influence of truncal vagotomy on canine gastric emptying of liquids. A. C. Springfield, C. O. Weddle II, H. S. Ormsbee III, R. F. Barreras and P. Bass, Am. J. Surg., 128, 678-681 (1974).

48. Effect of bile salts on partitioning and oral toxicity of the bisquaternary ammonium drug decamethonium bromide. T. S. Gaginella, J. H. Perrin, J. J. Vallner and P. Bass, J. Pharm. Sci., 63, 790-792, 1974.

49. Nicotine base permeation through silicone elastomers: comparison of dimethylpolysiloxane and trifluoropropylmethylpolysiloxane systems. T. S. Gaginella, P. G. Welling and P. Bass, J. Pharm. Sci., 63, 1849-1852 (1974).

50. Inhibitory actions of laxatives on motility and water and electrolyte transport in the gastrointestinal tract. J. J. Stewart, T. S. Gaginella, W. A. Olsen and P. Bass, J. Pharmacol. and Exp. Ther., 192, 458-467 (1975).

51. Inhibition of small intestinal mucosal and smooth muscle cell function by ricinoleic acid and other surfactants. T. S. Gaginella, J. J. Stewart, G. W. Gullikson, W. A. Olsen and P. Bass, Life Sciences, 16, 1595-1606 (1975).

52. Fatty acid inhibition of water absorption and energy production in the hamster jejunum. T. Gaginella, P. Bass, W. Olsen and A. Shug, FEBS Letters, 53, 347-350 (1975).

53. Actions of ricinoleic acid and structurally related fatty acids on the gastrointestinal tract. I. Effects on smooth muscle contractility in vitro. J. J.Stewart, T. S. Gaginella and P. Bass, J. Pharmacol. and Exp. Ther., 195, 347-354 (1975).

54. Actions of ricinoleic acid and structurally related fatty acids on the gastrointestinal tract. II. Effects on water and electrolyte absorption in vitro. T. S. Gaginella, J. J. Stewart, W. A. Olsen and P. Bass, J. Pharmacol. and Exp. Ther., 195, 355-361 (1975).

55. Effects of pyloroplasty on the electrical activity of the canine gastroduodenal junction. H. S. Ormsbee III, G. R. Mason, and P. Bass. Proceedings of the Fifth International Symposium in Gastrointestinal Motility, Typoff Press, Belgium, 1975, 293-299.

56. Effect of intravenous C-terminal octapeptide of cholecystokinin and intraduodenal ricinoleic acid on contractile activity of the dog intestine. J. J. Stewart and P. Bass, Soc. Exp. Biol. and Med., 152, 213-217 (1976).

57. The effects of sodium ricinoleate on small intestinal function and structure. W. S. Cline, V. Lorenzsonn, L. Benz, P. Bass and W. A. Olsen, J. Clin. Invest., 58, 380-390 (1976).

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